

speaking it shows that of 97 to 98 per cent of the cases who leave the hospital after nephrectomy for tuberculosis about 25 per cent die of urinary tuberculosis, and of this number one-half die within the first two years and the balance within five years. In such cases the author believes that they either had tuberculosis of the remaining kidney, though unsuspected, or they must have developed it very promptly after operation. There is no method now at our disposal which will enable us to diagnose "closed" tuberculosis, not the form in which closure of the ureter has resulted, but those tuberculous lesions of the kidney which are contained entirely within the renal parenchyma and communicate at no point with the renal pelvis. These cases undoubtedly exist and show a urine indistinguishable from the normal. As a result of his study of the problem the author believes that in a certain number of the cases we must expect tuberculosis of the remaining kidney as an unavoidable consequence of the operation, and he states there is nothing which we can do to avoid its occurrence, and it must therefore be charged off to depreciation. There is another possibility of infection occurring after operation, namely, infection of the remaining kidney as a consequence of the measures taken to arrive at an accurate diagnosis. Diagnosis of sufficient accuracy to warrant operation depends inevitably upon cystoscopy and ureteral catheterization. The majority of these patients have tuberculosis of the bladder secondary to their renal disease. In many of them the technical difficulty of the examination is great and trauma nearly or quite unavoidable, and were we dealing with any other form of infection of a unilateral type we should be gravely conscious of the danger of infecting the remaining sound kidney. This has been largely overlooked, and though it may be regarded as inevitable to accurate diagnosis, we must overhaul our methods and be sure that they expose the patients to the slightest possible danger of infection.

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## **PATHOLOGY AND BACTERIOLOGY**

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UNDER THE CHARGE OF

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**Experimental Rickets in Rats. I. A Diet Producing Rickets in White Rats and its Prevention by the Addition of an Inorganic Salt.**—**SHERMAN and PAPPENHEIMER** (*Jour. Exper. Med.*, 1921, xxxiv, 189) indicate advantages of using rats for the experimental study of rickets, calling attention to the close resemblance of the lesions to those of human rickets, the ease with which controls of the same litters can be obtained, the rapid development of the lesions, the elimination of

variations in susceptibility, the possibility of working with large numbers, the economy of space and expense and finally the easy manner in which histological examination of bones can be carried out. With no failures fifteen rats were rendered rachitic by a diet consisting of patent flour (95 per cent), calcium lactate (3 per cent), sodium chloride (2 per cent) and in some ferric citrate (0.1 per cent). The substitution of 0.4 per cent secondary potassium phosphate in the diet completely inhibited the development of rickets. Quantitative determination of calcium in the bodies of parallel rats showed a marked increase of calcium content in the rats receiving the added phosphate over those which developed rickets. "While it was shown by the roentgen rays, by histological examination and by quantitative chemical analysis that added potassium phosphate increased the assimilation and normal depositions of calcium, it may be the quantitative relationship between the inorganic ions rather than actual deficiency of any one of them which was here the determining factor in the cause or prevention of rickets." The authors state that their experiments and conclusions do not exclude the possibility of other causes of rickets than those discussed by them.

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**Note on the Preservation of Stock Strains of *Treponema Pallidum* and on the Demonstration of Infection in Rabbits.**—BROWN and PEARCE (*Jour. Exper. Med.*, 1921, xxxiv, 185) have been able to dispense with serial transfers of stock strains of *Treponema pallidum* by keeping a sufficient number of infected animals to guard against loss of the strain by their death. When it is desired to recover the organism a popliteal lymph node may be excised, aseptically, minced, ground in a mortar and an emulsion prepared by the addition of 1.5 cc sterile normal salt solution. One half cc of this emulsion is then injected into a testicle of one or more rabbits. The inoculation should be made six to eight weeks before the organism is needed. The same method is applicable for the demonstration of infection in experimental animals. The authors caution that atrophy of the testis may occur instead of the usual granulomatous enlargement and, in exceptional instances, infection may be recognized by the development of an adenopathy when no lesions can be detected at the site of inoculation. The method is based on the experimental evidence that there was a constant invasion and localization of the organism in the superficial lymph nodes, that the infection persisted indefinitely and that the organism could be recovered at any time from such nodes as the popliteals.

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**Bacteriologic Studies of the Upper Respiratory Passages.**—In a series of four articles PILOT and PEARLMAN (*Jour. Infect. Dis.*, 1921, xxix, pp. 47, 51, 55 and 62) report bacteriological investigations to determine the incidence of hemolytic streptococci in the adenoids and of pneumococci, non-hemolytic streptococci, influenza bacilli (Pfeiffer), diphtheria bacilli and diphtheroids in the tonsils and adenoids. From cultures of 25 nasopharyngeal swabs and the surfaces of 78 extirpated adenoids from children hemolytic streptococci were recovered in 55 per cent, while from the crypt-like depressions of the adenoids of the same persons they were found more abundantly in 61 per cent. The excised tonsils of the same persons revealed hemolytic streptococci in